A QUANTITATIVE ESTIMATE OF NONSMOKERS' LUNG CANCER RISK FROM PASSIVE SMOKING

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This work presents a quantitative assessment of nonsmokers' risk of lung cancer from passive smoking. The estimates given should be viewed as preliminary and subject to change as improved research becomes available. It is estimated that U.S. nonsmokers are exposed to from 0 to 14 mg of tobacco tar per day and that the typical nonsmoker is exposed to 1.4 mg per day. A phenomenological exposure-response relationship is derived, yielding 5 lung cancer deaths per year per 100,000 persons exposed, per mg daily tar exposure. This relationship yields lung cancer mortality rates and mortality ratios for a U.S. cohort which are consistent to within 5% with the results of both of the large prospective epidemiological studies of passive smoking and lung cancer in the United States and Japan. Aggregate exposure to ambient tobacco smoke is estimated to produce about 5000 lung cancer deaths per year in U.S. nonsmokers aged ≥ 35 yr, with an average loss of life expectancy of 17 ± 9 yr per fatality. The estimated risk to the most-exposed passive smokers appears to be comparable to that from pipe and cigar smoking. Mortality from passive smoking is estimated to be about two orders of magnitude higher than that estimated for carcinogens currently regulated as hazardous air pollutants under the federal Clean Air Act.

Introduction

Exposure of nonsmokers to indoor air pollution from tobacco smoke (also known as involuntary or passive smoking) has recently become a public health concern (USSG, 1982) for several reasons: such exposure is widespread (Repace and Lowrey, 1980; Friedman *et al.*, 1983); studies of the effects of tobacco smoke on smokers worldwide have implicated it as the most important cause of lung cancer (USSG, 1982; Doll and Peto, 1981); existence of a threshold for carcinogenesis is doubtful (USSG 1982; IRLG, 1979; U.S. EPA, 1979a; IARC, 1979; Pitot, 1981); and there is suggestive new evidence of lung cancer (and other serious health effects) in nonsmokers exposed to ambient concentrations of tobacco smoke (Trichopoulos, 1981, 1983; Hirayama, 1981a, 1981b, 1983a, 1983b; Garfinkel, 1981; Correa *et al.*, 1983; Knoth *et al.*, 1983; Gillis *et al.*, 1983; Koo *et al.*, 1983; Kabat and Wynder, 1984; Miller, 1984; Sandler *et al.*, in press a,b).

There are three important fractions of tobacco smoke: mainstream smoke, which the smoker inhales directly into the lung; exhaled mainstream smoke, that fraction of the mainstream smoke which is not retained in the lungs of the smoker, and sidestream smoke, that fraction of tobacco smoke emanating directly from the burning end of the cigarette into the air. Nonsmokers are commonly exposed to tobacco combustion products in diluted sidestream and exhaled mainstream tobacco smoke from cigarettes, cigars, and pipes (Repace and Lowrey, 1980). Tobacco smoke contains 60 known or suspect carcinogens, including 51 in the phase containing particulate matter; the carcinogenic activity of tobacco smoke appears to require this phase (USSG, 1982). Animal bioassays indicate that sidestream tobacco tar is more carcinogenic per unit weight than mainstream tar (Wynder and Hoffman, 1967). For public health purposes, it will be assumed that mainstream and sidestream smoke have similar human carcinogenic potency.

In his 1982 report on cancer and smoking (USSG, 1982), the U.S. Surgeon General asserted that despite the incompleteness of the evidence, nonsmokers should

*The views presented in this article are those of the authors, and do not necessarily reflect the policies of their respective agencies.

avoid exposure to second-hand smoke to the extent possible, a risk-management judgement supported by the World Health Organization and the National Academy of Sciences (WHO, 1979; NRC, 1981).

This raises the question of whether the quantity of tobacco tar to which the average nonsmoker is exposed creates a significant risk of lung cancer. In order to answer this question, a quantitative risk assessment is first justified and then performed. Risk assessment is the use of science to define the health effects of exposure of individuals or populations to hazardous materials or situations (NRC, 1983). Risk assessments contain some or all of the following four steps:

(1) Hazard identification—the determination of whether a particular chemical is or is not causally linked to certain health effects.

(2) Dose-response assessment—the determination of the relation between the magnitude of exposure and the probability of occurrence of the health effects in question.

(3) Exposure assessment—the determination of the extent of human exposure before or after application of regulatory controls.

(4) Risk characterization—the description of the nature and often the magnitude of the human risk, including attendant uncertainty.

In other words, quantitative risk assessment deals with the question of how much morbidity and mortality an agent is likely to produce given specified levels of exposure. Typically utilized in the regulation of carcinogens, it is important because control efforts cannot proceed without assurance that the health gains are worth the cost (Lave, 1983; Albert, 1983). On the basis of such assessments, informed risk management judgements can be made.

This work draws upon the epidemiology of lung cancer (USSG, 1982; Pitot, 1981; USSG, 1979; Ives, 1983) and on indoor air pollution physics (Repace and Lowrey, 1980, 1982; NRC, 1981) to produce a risk analysis (IRLG, 1979; U.S. EPA, 1979a; Lave, 1983; COST, 1983; Fischoff *et al.*, 1981; NRC, 1983) in which nonsmokers' lifestyles are correlated to exposure to airborne tobacco tar, and incidence of lung cancer. This analysis first reviews estimates of the average exposure of the general population of ambient tobacco smoke. Second, it reviews studies linking tobacco-related disease in nonsmokers to exposure-related variations in lifestyle. Third, it couples these two factors to develop a phenomenological estimate for the aggregate lung cancer risk to the U.S. nonsmoking population, and develops an exposure-response relationship for the estimation of the risk to the most-exposed. Fourth, it compares the estimated level of lung cancer mortality and resultant loss of life expectancy from passive smoking to those from cigarette, pipe, and cigar smoking. Fifth, it compares the predictions of alternate exposure-response relationships with the results of two large prospective epidemiologic studies of passive smoking and lung cancer, and performs a sensitivity analysis. Finally this work compares the estimated risk from ambient tobacco smoke to that from various airborne carcinogens currently being regulated in the United States as hazardous air pollutants, to place the significance of the estimated risk in perspective.

Variation of Exposure with Lifestyle

In earlier work (Repace and Lowrey, 1980, 1982, 1983, 1984; Repace, 1981, 1982, 1983, 1984, in press; Repace *et al.*, 1980, 1984; Bock *et al.*, 1982) factors affecting nonsmokers' exposures to tobacco smoke were studied, and field surveys of the levels of respirable particles were conducted indoors and out, in both smoke-free and smoky environments. This work established that ambient tobacco smoke imposed significant air pollution burdens on nonsmokers, and, using controlled experiments (Repace and Lowrey, 1980, 1982, 1983), a model was developed to estimate those exposures. This model predicts that the exposure of U.S. nonsmokers ranges from 0 to 14 mg of cigarette tar per day (mg/d), depending upon the nonsmokers's lifestyle. As derived in Appendix A and shown in Table 1, the average population exposure for adults of working age, averaging over the work and home microenvironments, is about 1.43 mg/day (Repace and Lowrey, 1983) with an 86% exposure probability.

Table 1, derived from the model in Appendix A, estimates probability-weighted exposure to the particulate phase of ambient tobacco smoke for a typical U.S. adult nonsmoker. Exposures received in other (Repace *et al.*, 1980) indoor microenvironments, outdoors, and in transit, which account for the remaining 12% of people's time, were omitted. Table 1 is derived from considerations that ambient concentrations of tobacco tar have been found to be directly proportional to the smoker density and inversely proportional to the ventilation rate (Repace and Lowrey, 1980). Ventilation rate tables given by ASHRAE (1981), can be used to estimate both the range in ventilation rate (from the design mechanical rates) and smoker density (from the design occupancies), and thus upper and lower bounds and average concentrations for model workplace and home microenvironments can be estimated.

Table 1 suggests that individuals receiving exposure both at home and at work constitute a high exposure group, with the workplace appearing four times as strong a source of exposure as the home; the reason for this differential is the generally higher occupancy (i.e., smoker density) encountered in the workplace (Repace and Lowrey, 1982; ASHRAE, 1981). This estimate of exposures represents a modeled weighted average taken over the entire population, including those who are not exposed.

Table 1. Estimated probabilities of nonsmokers' exposure to tobacco smoke at home and at work (after Repace and Lowrey, 1983; Appendix A).[a] Nonexclusive probability of being exposed at work: 63%; probability of *not* being exposed at work: 37%. Nonexclusive probability of being exposed at home: 62%; probability of *not* being exposed at home: 38%.

Lifestyle: Daily Average Probability of Being Exposed (Rounded Values)		Exposure (mg) Modeled Daily Average	Exposure (mg) Daily Probability-Weighted
At work and at home: %	63 x 62 = 39	2.27	0.89
Neither at work nor at home: %	37 x 38 = 14	0.00	0.00
At home but not at work: %	62 x 37 = 23	0.45	0.10
At work but not at home: %	63 x 38 = 24	1.82	0.44
Total: %	100		1.43

[a]The estimated exposure to the particulate phase of ambient tobacco smoke for U.S. adults of working age, at work and at home (these two microenvironments account for an estimated 88% of the average person's—both smokers and nonsmokers—time), determined from average concentrations of tobacco smoke calculated for model workplace and home microenvironments, weighted for average occupancy, as derived in Appendix A.

Jarvis and Russell (in press), in a study of urinary cotinine (a nicotine metabolite) in a sample of 121 self-reported nonsmokers, state that only 12% of subjects had undetectable cotinine levels, despite nearly 50% reporting no passive smoke exposure. In a study of 472 nonsmokers, Matsukura (1984) examined the relationship of urinary cotinine to the smokiness of their environment, and found that nonsmokers who lived or worked with smokers had higher cotinine levels than those who did not. Matsukura *et al.*, (1984) also found that cotinine levels increased with the number of smokers present in the home and the workplace; however, none of the differences were statistically significant, except for the lowest urinary cotinine level of the nonsmokers who were not exposed to tobacco smoke in the home or the workplace. These studies respectively illustrate the widespread exposure of nonsmokers to ambient tobacco smoke, and the relative importance of the domestic and workplace microenvironments in such exposures.

Epidemiological Evidence for the Variation of Risk with Lifestyle: Pulmonary Effects

White and Froeb (1980) evaluated the effect of various degrees of long-term (>20 yr) workplace exposure to tobacco smoke on 2100 healthy middle-aged workers. Of these workers, 83% held professional, managerial, or technical positions, while the remaining 17% were blue-collar workers. Relative to those not exposed at home or at work, passive smokers of both sexes suffered statistically significant declines in mid- and end-expiratory flow rates which averaged about 13.5% and 22% respectively, and did not differ significantly from the values measured in nonsmoking or light smokers of cigarettes, pipes, and cigars. They concluded that chronic exposure to tobacco smoke in the work environment reduces small airways function to the same extent as smoking 1 to 10 cigarettes per day.

Kauffmann *et al.* (1983) compared pulmonary function in about 3800 people in France: 849 male "true" nonsmokers (defined as those not exposed at home), 165 male passive smokers (defined as those exposed at home), 826 female "true" nonsmokers, and 1941 female passive smokers. The authors restricted the analysis to subjects aged 40 yr or older (i.e., to those who had been exposed for 15 or more years to smoking by their spouses) and who were living in households with no persons over the age of 18 yr except their spouses. They found that nonsmoking subjects of either sex whose spouses were current smokers of at least 10 g (about 10 cigarettes) of tobacco a day had mid-expiratory flow rates averaging 11.5% lower than those married to nonsmokers. For women in social classes with the highest percentage of paid work, the effect of workplace smoking appeared to confound the effect of passive smoking at home. However, in the large subgroup of women without paid work (i.e., not exposed to workplace smoking), a clear dose-response relationship to amount of husbands' smoking was observed. They concluded that women living with heavy smokers appeared to have the same reductions in mid-expiratory flow rates as light smokers, and that after 15 yr exposure in the home environment, passive smoking reduces pulmonary function.

A third study by Kasuga (1983) of urinary hydroxyproline-to-creatinine (HOP-r) ratios as a function of passive smoking status showed that HOP-r levels in nonsmoking wives and children varied in a dose-response relationship with husbands' and parental smoking habits, when adjusted for pre-existing respiratory disease. Kasuga (1983) asserts that HOP-r serves as a marker to detect deleterious active and passive smoking effects on the lung, before and after the manifestation of clinical symptoms, and that urinary HOP-r in light-smoking women is almost equivalent to HOP-r in nonsmoking wives with heavy-smoking husbands.

These three epidemiologic studies provide evidence that variations in the exposure of adult nonsmokers to ambient tobacco smoke at home and, particularly, at

work, can produce observable pulmonary effects. Like effects have been observed in children exposed at home (Tager *et al.*, 1983).

Cancer

Thirteen epidemiologic studies have explicitly examined the lung cancer risk incurred by the nonsmoking spouses of cigarette smokers. In all but one study, the only exposure variable was the strength of the spouse's smoking habit. The studies were conducted in Greece (Trichopoulos *et al.*, 1981, 1983), Japan (Hirayama, 1981a, 1981b, 1983a, 1983b), the United States (Garfinkel, 1981; Correa, *et al.*, 1983; Kabat and Wynder, 1984; Miller, 1984; Sandler, *et al.*, a and b, in press), Germany (Knoth *et al.*, 1983), Scotland (Gillis *et al.*, 1983), and Hong Kong (Chan and Fung, 1982; Koo *et al.*, 1983).

In the Greek study, Trichopoulos *et al.* (1981, 1983) used the case-control technique: involuntary exposure to cigarette smoke as measured by the husbands' daily consumption was found to increase the average risk of lung cancer by a factor of 2.4 ($p < 0.01$) when 77 lung cancer patients were compared to 225 controls, and a dose-response relationship was observed. Divorce, remarriage, husband's death, and change in smoking habits were considered.

In the Japanese study (from 1966 to 1981) of lung cancer in 91,540 nonsmoking women, Hirayama (1981a, 1981b, 1983a, 1983b) used the prospective technique: relative to those women not exposed at home (controls), involuntary exposure of wives of smokers was found to increase the average risk of lung cancer by a factor of 1.78 ($p < 0.001$), where the exposure was also estimated from husbands' daily consumption. The annual lung cancer death (LCD) rate in the controls was 8.7 per 100,000. Hirayama found that the exposed wives experienced an average annual increase in lung cancer mortality rate of 6.8 per 100,000, with a range of 5.3 to 9.4 per 100,000, in a dose-response relationship depending upon the degree of the husband's smoking. Hirayama found further that the risk of lung cancer death in nonsmoking women increased both with the time of exposure and number of cigarettes smoked daily by the husband. Hirayama also reported a factor of 2.9 (± 0.3, at the 95% confidence level) for increased risk of lung cancer in 1010 nonsmoking husbands with smoking wives.

More recently, Hirayama extended his earlier work to suggest increased risk of nasal sinus cancer, and ischemic heart disease in passive smokers, and evidence of decreased lung cancer risk in nonsmoking wives of exsmokers. With respect to cancer of the para-nasal sinuses in nonsmoking wives ($n = 28$), Hirayama found standardized mortality ratios of 1.00, 2.27, 2.56, and 3.44 when husbands were nonsmokers, smokers of 1–14, 15–19, and >20 cigarettes per day, respectively ($p = 0.01$). For ischemic heart disease, risk elevations for nonsmoking wives ($n = 494$) with the extent of husbands' smoking were reported, with standardized mortality ratios of 1.00, 1.10, and 1.31 when husbands were nonsmokers, smokers of 1–19, and >20 cigarettes/day, respectively ($p < 0.02$). For lung cancer, the standardized mortality ratio of lung cancer in nonsmoking women ($n = 200$) was 1.00, 1.36, 1.42, 1.58, and 1.91 when husbands were nonsmokers, exsmokers, daily smokers of 1–14, 15–19, and >20 cigarettes/day, respectively.

In the first U.S. study, Garfinkel (1981) reported results from an analysis of data collected from the American Cancer Society's (ACS) prospective study of lung cancer risk in 176,739 nonsmoking white women (1960 to 1972) as a function of involuntary exposure as indicated by their husbands' cigarette consumption. Of the total, 72% of the nonsmoking women were married to smokers. Three smoking categories were identified: none, less than one pack (20 cigarettes) per day, or greater than one pack per day. Garfinkel reported statistically insignificant risk ratios of 1.00, 1.27, and 1.10, respectively, for the three categories (average 1.20 over the exposed categories). Also reported were age-standardized death rates, which were respectively 13.8, 12.9, and 13.1 lung cancer deaths per 100,000 person-yr for this cohort in 1960–1964, 1964–1968, and 1968–1972 (average 13.3 per 100,000 person-yr for the period 1960–1972). The death rates were standardized to the distribution of white men and women combined for the U.S. population in 1965, which decreased the rates for females "slightly."

More recently, Correa *et al.* (1983) studied 8 male and 22 female nonsmoking lung cancer cases and 180 male and 133 female controls as part of a larger study including smokers, with 1338 lung cancer cases and 1393 controls, in Louisiana. They reported that nonsmokers married to heavy smokers had an increased risk of lung cancer, as did smokers whose mothers smoked. Men with smoking wives had a nonsignificant risk ratio of 2.0 compared to their counterparts with nonsmoking wives, and women with smoking husbands had an average risk ratio of 2.07 ($p < 0.05$) compared to women with nonsmoking husbands. An exposure-response relationship was observed, with the peak risk reaching 3.5 ($p < 0.05$). The combined data for men and women passive smokers was significant ($p < 0.05$) for the heavier smoking category (≥ 41 pack-years).

A third U.S. case-control study, by Kabat and Wynder (1984), reported on passive smoking and lung cancer in nonsmokers for 25 male cases and 25 controls, and 53 female cases and 53 controls, where the majority of the patients were from New York City. The controls consisted of patients hospitalized for non-smoking-related diseases, roughly two-thirds being cancer patients. No differences on exposure to passive smoking at home or at work were found in the women. However,

the male passive smokers displayed a statistically significant ($p = 0.05$) difference in lung cancer (odds ratio 1.6) relative to the non-exposed group.

A fourth U.S. study by Miller (1984) of mortality from all forms of cancer in 123 nonsmoking women (only 5 lung cancer cases) as a function of husband's smoking history reported a nonsignificant odds ratio of 1.4 for all women ($p = 0.15$) for women whose husbands smoked relative to those who did not; when employed women were excluded, the odds ratio increased to 1.94 and was statistically significant ($p < 0.02$).

A fifth U.S. study of Sandler *et al.* (in press a) also examined mortality from all forms of cancer related to passive smoking, in both nonsmokers and smokers [231 cases and 235 controls (70% white and 67% female); only 2 cases of lung cancer in nonsmokers] as a function of spouses' smoking habits. Cancer risk—adjusted odds ratio—(lung, breast, cervix, and endocrine) among individuals ever married to smokers was 2.0 times that among those never married to smokers ($p < 0.01$). This increased risk was not explained by confounding individual smoking habits, demographic characteristics, or social class.

In a sixth U.S. study, Sandler *et al.* (in press b) examined cancer risk in adulthood in 197 cases and 223 controls, 66% female, from early life exposure to parents' smoking. They found that mothers' and fathers' smoking were both associated with risk for hematopoietic cancers (Hodgkin's disease, lymphomas, and leukemias), and a dose-response relationship was seen for the latter two. The odds ratio for hematopoietic cancers increased from 1.7 when one parent smoked, to 4.6 when both smoked ($p < 0.001$).

In the first of two studies from Hong Kong, Chan and Fung (1982) found a lower incidence of passive smoking among 34 female lung cancer cases (40.5%) than among 66 female controls (47.5%). All patients and controls were interviewed concerning their smoking habits and those of their spouses, their cooking habits, including types of cooking fuel used. Histological diagnoses of tumors were obtained. Controls were taken from orthopedic patients.

In the second Hong Kong study, Koo *et al.* (1983) studied passive smoking in 56 female lung cancer cases and 85 female controls. Passive smoking cases had an excess of 3.8 yr of passive smoking (workplace plus domestic exposures) compared with controls, but the differences were not statistically significant ($p \leq 0.069$). However, among a subgroup of 8 marine dwellers, cases had 11.8 years more exposure than controls ($p = 0.0003$).

Knoth *et al.* (1983) reported on a study of 39 nonsmoking German females with lung cancer. 61.5% were found to have smoking spouses. The authors state that this percentage was threefold that expected on the basis of smoking habits of German males.

Gillis *et al.* (1984) reported preliminary results of a study of passive smoking and lung cancer in 91 male controls ($n = 2$) [the numbers in parentheses give the numbers of cases] without domestic passive smoking and in 90 subjects exposed at home ($n = 4$), and in 40 female controls ($n = 2$) and 58 subjects ($n = 6$). No effects of lung cancer were noted in the females, but elevated rates of myocardial infarction were reported (risk ratio 3.0). In the males, elevated rates of both lung cancer (risk ratio 3.25) and myocardial infarction (risk ratio 1.45) were reported. Gillis *et al.* state that since insufficient time has elapsed since the beginning of this study, no firm conclusions can be drawn relating to the incidence of cancer or other diseases.

Thus there are now a large number of studies providing evidence for increased risk of lung cancer from increased exposure to passive smoking. It might be expected that subgroups of the population which proscribe smoking among their membership would have a lower probability of passive smoking, and therefore should also have a lower incidence of smoking-related disease than the general nonsmoking population.

One such subgroup is the Church of Jesus Christ of the Latter Day Saints, popularly known as the Mormon Church, which advises against the use of tobacco. Enstrom (1978) found that active Mormons who were nonsmokers had standardized mortality rates for lung cancer which were 21% of those in the general population which includes smokers. This rate was found comparable to the rate of 19% for a sample of the U.S. general population "who had never smoked cigarettes." Interestingly, however, this result occurred despite the fact that 31% of the active Mormon cohort were former smokers. This confounding factor was not present for certain subgroups in the following study.

Phillips *et al.* (1980a, 1980b) have studied mortality (from 1960 to 1976) in Seventh Day Adventists (SDAs), a religious group that also follows rigorous proscriptions against the use of tobacco. As with the Mormons, SDAs have rates of mortality from lung cancer and other smoking related cancers that are fractions, 21% and 66%, respectively, of the rates for a demographically comparable group in the general U.S. population (including smokers) (1980a). Many SDAs work for church-run businesses. Thus, SDAs appear to be less likely than the general population to be involuntarily exposed to tobacco smoke, as children or as adults, at home or in the workplace, because neither SDA homes nor SDA businesses are likely to be places where smoking is permitted, and because the great majority of SDA family and social contacts are among other SDAs who do not smoke (See Appendix C).

Phillips *et al.* (1980a, 1980b) compared mortality in two demographically similar groups of Southern Californians: SDAs (from 1960 to 1976) and non-SDAs (from 1960 to 1971). A sizable subgroup (35%) of SDAs report prior cigarette use, especially among men (1980b). However, for two select subgroups of each group, 25,264 SDAs and 50,216 non-SDAs who were self-reported

Table 2. Age-adjusted SDA-to-non-SDA ratio of lung cancer mortality (after Phillips *et al.* (1980b).*

		By Health Habit Index		
	Average	Best Third	Average Third	Worst Third
I. All SDAs	0.54	0.54	0.40	0.96
II. SDAs who never smoked	0.41	0.41	0.32	0.78

Values shown are adjusted by Mantel-Haenzel procedure ($p \leq 0.01$).

*Lung cancer mortality ratios taken from a prospective study of two demographically similar cohorts. The non-SDA come from the general south California population, and were self-reported nonsmokers who never smoked. The SDA come from a southern California subgroup less likely to engage in passive smoking by virtue of lifestyle differences. The health habit index is a measure of how faithfully individuals adhered to the Church's teachings; the worst third were also more likely to have a non-SDA spouse. (Values quoted in text are the reciprocals of numbers given here.) Phillips *et al.* (1980a, 1980b) reported results for all SDA, and reported replicating these data for SDA who never smoked, as shown. The SDA subjects and non-SDA subjects for this study consisted of white California respondents to the same four-page self-administered questionnaire collected by the American Cancer Society study of 1 million subjects throughout the United States (NCI, 1966; Garfinkel, 1981; Phillips *et al.*, 1980a, 1980b).

nonsmokers who had never smoked, age-adjusted mortality rates were compared for smoking-related and nonsmoking-related diseases. Table 2 compares age-adjusted lung cancer mortality ratios for two SDA cohorts relative to nonsmokers in the general population who never smoked. The first cohort consists of all SDA, and includes those who never smoked, exsmokers, and smokers. The first row of Table 2 gives the mortality ratios relative to the never-smoked non-SDAs in the general population. The second row compares the second SDA cohort (those who never smoked) to the non-SDA who never smoked. The values given are averaged over both sexes. From Table 2 the results show that the non-SDA group of nonsmokers who had never smoked (but who were more likely to suffer involuntary exposure to tobacco smoke) had an average lung cancer mortality rate of 2.4 times that of the never-smoked SDAs (the group less likely to have suffered such exposure by virtue of their lifestyle). This concludes the review of evidence relating variations of lifestyle to variations in lung cancer risk in nonsmokers.

Does Ambient Tobacco Smoke Pose a Carcinogenic Hazard?

The International Agency For Research on Cancer (IARC) criteria for causality to be inferred between exposure and human cancer state that confidence in causality increases when (1) independent studies agree; (2) associations are strong; (3) dose-response relationships exist, and (4) reduction in exposure is followed by reduction in cancer incidence (IARC, 1979). These criteria are applied here as follows:

1. There are now 14 studies, covering 6 cultures, indicating a relationship between exposure to ambient tobacco smoke and incidence of lung cancer. If the studies are divided into substudies of men and women, this yields 20 substudies, all but 2 of which suggested an increased cancer mortality from passive smoking, and 12 of which attained statistical significance. Moreover, the mortality ratios based on spouses' smoking as an exposure variable, cluster around the value 2.0. Thus, many independent studies agree.

2. Mainstream tobacco smoke is strongly associated with lung cancer. The U.S. Surgeon General (USSG, 1982) asserts that mainstream cigarette smoke is a major cause of cancers of the lung, larynx, oral cavity, and esophagus, and is a contributory factor for the development of cancers of the bladder, pancreas, and kidney, where the term contributory factor does not exclude the possibility of causality. Both smokers and nonsmokers are exposed to exhaled mainstream and sidestream tobacco smoke. Sidestream smoke by animal bioassay has been found to be of greater potency than mainstream smoke.

3. Five of the 14 studies reported dose-reponse relationships between passive smoking and lung cancer. Dose-response relationships between lung cancer and active cigarette smoking show increasing mortality with increasing dosage of smoke exposure, and an inverse relationship to age of initiation (USSG, 1982). Dose-response relationships are also shown for smokers whose smoking habits are like heavy passive smoking (Wynder and Goodman, 1983; Jarvis and Russell, in press), i.e., in cigarette smokers who do not inhale, and in pipe and cigar smokers, who also are unlikely to inhale (USSG, 1982; USSG, 1979).

4. Reductions in lung cancer incidence for reduction in exposure have been found in all major studies of active smoking (USSG, 1982). The one study of passive smoking and lung cancer which examined this question also found a similar result (Hirayama, 1983b). Furthermore, the comparison of the SDAs who never smoked, and who should have reduced exposure relative to the non-SDAs who never smoked, also appears to exhibit this effect.

On the basis of the IARC criteria, the evidence appears to be sufficient for reasonable anticipation of an increase in lung cancer mortality from passive smoking, justifying a quantitative risk assessment. The significance of the public health risk will now be estimated.

Estimation of Total LCD Risk and a Phenomenological Exposure-Response Relationship

A phenomenological exposure-response relationship is now derived based on consistency (Hirayama, 1983b)

of evidence provided by studies of lung cancer in nonsmokers and from our exposure assessment. The Seventh Day Adventist Study by Phillips *et al.* (1980a, 1980b) appears to provide the best evidence of the magnitude of the lung cancer effect from passive smoking among U.S. nonsmokers.

A calculation (Appendix C) based on the age-standardized differences in lung cancer mortality rates between SDAs who never smoked and demographically comparable non-SDAs who never smoked (age groups 35 to 85+) from the studies of Phillips *et al.* (1980a, 1980b) yields an estimated 4700 lung cancer deaths (LCDs) for the 62.4 million U.S. nonsmokers (USDC, 1980) at risk (USSG, 1979) aged ≥ 35 yr. This in turn yields a passive smoking risk rate of 7.4 LCDs per 100,000 person-yr (4700 LCDs/yr per 62,424,000 persons), in good agreement with the value of 6.8 per 100,000 person-yr reported in the Hirayama (1981) study. To place the estimated mortality in perspective, 4700 deaths was about 5% of the total annual LCDs, and about 30% of the LCDs in nonsmokers in 1982 (USSG, 1982).

The exposure of nonsmokers in the U.S. population of working age, taken from the model results in Table 1, appears to be a weighted average of about 1.43 mg of tobacco tar per day, including the estimated 14% of the population who received no exposure at home or work. The carcinogenic risks will be assumed to apply even to retired persons, whose exposures are reported to be less than the employed (Friedman *et al.*, 1983), because the risks of lung cancer from smoking decline only slowly even with total cessation of exposure (USSG, 1982), and because the risks of lung cancer increase exponentially with age (NCI, 1966).

Using the statistical risk of 7.4 LCDs per 100,000, and dividing by the average exposure of 1.43 mg/day, we estimate a phenomenological exposure-response relationship appropriate for the general U.S. population at risk of about 5 LCDs per 100,000 person-yr at risk per 1 mg/day nominal exposure.

The range in nominal exposure has been estimated to be 0–14 mg/day (Repace and Lowrey, 1980). Studies of lung cancer and passive smoking across three cultures have shown an exposure-response relationship. Thus, the assumption of an exposure-response relationship is justified, and a linear exposure-response function (Doll and Peto, 1981; IRLG, 1979; U.S. EPA, 1979; Crump *et al.*, 1976) is assumed. With zero excess risk from tobacco smoke for zero exposure, and applying the exposure-response relationship derived above, with the maximum exposure of 14 mg/day, a maximum risk of about (14 × 5) = 70 LCDs per 100,000 person-yr is estimated for the most-exposed lifestyle. This lifestyle has been previously typified by that of a nonsmoking musician who performs regularly in a smoky nightclub and who resides in a small apartment with a chainsmoker; many other scenarios may be drawn (Repace and Lowrey, 1980).

Estimated Loss of Life Expectancy

Reif (1981a, 1981b) argues that there exists a genetically determined distribution in natural susceptibility to lung cancer in people; the effect of exposure to tobacco smoke is to shift this distribution toward death at earlier ages. In other words, exposure to tobacco smoke produces a loss of life expectancy. One method of presenting risk data involves calculation of the loss of life expectancy, in units of days of life lost per individual, averaged over the entire population at risk. When the average life-loss is multiplied by the number of individuals at risk, the impact of the hazard on society in person-yr of life lost can be assessed. More important, one can display the age-specific probabilities of death from the hazard, as well as the average number of years of life lost by the average victim. Appendix C gives the method of calculation.

Averaged over all of the population at risk, (i.e., including those who die of other causes), the average loss of life expectancy from passive smoking is calculated (appendix C) to be 15 days, which is equivalent to an ultimate loss of 2.5 million person-yr of life for the total at-risk U.S. population in 1979 over 35 yr of age (62.4 million persons). The estimated worst-case loss of life expectancy is 148 days, again averaged over all of the population at risk. The estimated mean life expectancy lost by a passive-smoking lung cancer *victim* is 17 ± 9 yr.

How does the calculated average loss of life expectancy for heavy passive smoking compare with the average loss of life expectancy found in active smokers? The modeled worst-case lifestyle might be reasonably expected to have lesser exposure, and hence lesser risk than active smokers. Table 3, adapted from Cohen and Lee (1979), gives this comparison. The estimated most-exposed lifestyle has about ⅔ the loss of life expectancy of the average pipe smoker, about ½ the loss of the

Table 3. Estimated loss of life expectancy from active smoking (all causes) and passive smoking (lung cancer only), adapted from Cohen and Lee (1979).

Cause	Days
Cigarette smoking – male	2250
Cigarette smoking – female	800
Cigar smoking	330
Pipe smoking	220
Passive Smoking[a] (est. most exposed lifestyle)	148
Passive smoking[a] (est. average lifestyle)	15

[a]Estimated this work (see Appendix C); averaged over all nonsmokers at risk, i.e., those who are presumed to die from passive smoking-induced lung cancer, and those who do not. Estimates given for passive smoking are phenomenological estimates.

average cigar smoker, and 1/10 of that for active cigarette smoking.

Estimate of an Exposure-Response Relationship Based on Risks in Smokers

An alternative extrapolated exposure-response relationship is now derived from evidence provided by studies of lung cancer in cigarette smokers. Using the Surgeon General's estimate that 85% of all lung cancers are due to smoking (USSG, 1982), a current annual LCD rate to smokers at risk of about 316 per 100,000 is estimated (see Appendix B). Assuming a one-hit model (see Appendix B) for extrapolation of the risk (which in this range is functionally equivalent to the linear assumption that a milligram of tobacco tar inhaled by a nonsmoker produces a response equivalent to that in a smoker) yields an estimate of about 0.87 LCDs/100,000 person-yr. This corresponds to an exposure-response relationship of 0.6 LCDs/ 100,000 person-yr per mg/day, and an annual aggregate risk estimate of about 555 LCDs per year, an order of magnitude lower than the phenomenological estimate.

Discussion of Alternative Exposure-Response Relationships

We now speculate on why these two different methods produce such disparate estimates of risk. One possibility is that nonsmokers may have a reduced tolerance to the effects of tobacco smoke. Another possibility is a "large dose" effect (Jarvis and Russell, in press), whereby exposure to tobacco tar at the lesser doses experienced by nonsmokers produces a greater risk per unit dose than the greater doses experienced by active smokers, whose lung tissue is saturated by carcinogenic tar. Large dose effects have been observed in cancer induction by ionizing radiation, in which the dose-response curve has a linear form at low doses, a quadratic upward (positive) curvature at intermediate doses, but a downward (negative) curvature at high doses (NRC, 1980). Downturns in exposure-response curves of lung cancer in smokers of more than 40 cigarettes per day have been observed by Doll and Peto (1978) and Hirayama (1974). The effect of a leveling-off or downturn in the exposure-response curve at large exposures would be to cause a linear model to underestimate the risk when extrapolated (Hoel *et al.*, 1975, 1983; NRC, 1980) over two orders of magnitude to low exposures.

A third possibility is generated by modeling the dose, as opposed to the exposure, of nonsmokers to tobacco smoke. Nonsmokers' exposure is translated into dose by means of a simple single-compartment model for lung deposition and clearance (Repace, 1983). This model suggests that tar may accumulate on the surface of nonsmokers' lungs to an equilibrium dose an order of magnitude higher than the nominal exposure, to a level of about 16 mg/day, due to the long pulmonary residence times for respirable aerosols. If this 16-mg dose, rather than the 1.4-mg exposure, is the operative factor, then the typical passive smoker would have a risk, according to the one-hit model, of about 9 per 100,000, in agreement with the phenomenological estimate.

There is support for this argument from Matsukura's study (1984), which showed that heavy passive smokers had urinary cotinine levels comparable to active smokers of less than 3 cigarettes per day, and from Kasuga's study (1983), which also showed that heavy passive smokers had urinary hydroxyproline levels almost equivalent to that of light smokers. Moreover, similar observations have been found indicating that serum thiocyanate (Cohen and Bartsch, 1980) and benzpyrene (Repetto and Martinez, 1974) levels in some passive smokers were comparable to the elevated levels typically found in smokers.

The simple model we have proposed ignores the effect of cancer latency. The long latency period for lung cancer indicates that childhood passive smoking may be an important factor affecting risk in adult life: Doll and Peto (1981) have suggested that the effect of passive smoking may be surprisingly large because lifelong exposure may produce a lung-cancer effect four times as great as that which is limited to adult life (recall the observation of Sandler *et al.*, in press: childhood passive smoking appeared to elevate the cancer risk of adults). As Bonham and Wilson (1981) have shown from a national study of 40,000 children in 1970, 62% came from homes with one or more smokers, indicating that many adults receive exposure during childhood.

Sensitivity Analysis

Which of the two exposure-response relationships derived is more useful in explaining actual epidemiological data? The Garfinkel (1981) American Cancer Society (ACS) study of passive smoking and lung cancer, which spanned the years 1960 to 1972, reported a standardized mortality ratio of 1.20 and an annual lung cancer rate of 13.3 per 100,000 person-yr. Of the 176,739 women in the Garfinkel study, 28% had nonsmoking husbands. Thus, the "controls" numbered 49,487 and the total "exposed" were 127,252. According to census data (U.S. Dept. of Commerce, 1980), female participation rates in the labor force ranged from 37.1% in 1960 to 38.8% in 1965, 42.8% in 1970, and 43.7% in 1975, and were about 80% of the 1965 level in 1947. Thus, it appears that about 38% of the women in this study were in the labor force, and presumably exposed to passive smoking while at work. It is assumed that for both groups of women, control and exposed, 38% were employed and exposed to ambient tobacco smoke while at work.

As indicated in Table 1, typical U.S. nonsmoking

Group	Number
Total cohort	176,739
"True" controls: do not work, husbands do not smoke	30,682
"Tainted" controls: work, husbands do not smoke	18,805
Total "controls"	49,487
"Exposed" workers: work, husbands smoke	48,356
"Exposed" nonworkers: do not work, husbands smoke	78,896
Total "exposed"	127,252

adults are estimated to inhale 1.82 mg of tobacco tar per average day at work and 0.45 mg per average day at home, an exposure ratio of 4:1. This occurs because, although domestic and workplace air exchange rates are similar (Appendix A), workplace smoker densities tend to be far higher. Let the assumed basal rate of lung cancer deaths in these women from causes other than passive smoking be 8.7 per 100,000 (the age-adjusted rate for nonsmoking women married to nonsmokers in Hirayama's study, 1981a). The Garfinkel (1981) ACS cohort can now be broken down as shown in Table 4.

The Garfinkel (1981) study can be analyzed as follows, using the phenomenological exposure-response relationship of 5 LCDs/100,000 person-yr-mg/day.

The lung cancer deaths per 100,000 contributed by passive smoking are then 2.25 (0.45 × 5) for the home and 9.10 (1.82 × 5) for the workplace. Application of these figures to the numbers of true and tainted controls and working and nonworking exposed women yields, after addition of the basal risk of 8.7 per 100,000, the estimated rates for lung cancer deaths per 100,000 person-yr, as shown in Table 5. The ratio of risks (all exposed:all controls) is thus 1.19. The ratio (averaged over husbands' heavy and light smoking categories) in the Garfinkel (1981) study was 1.20, less than a 1% difference. The lung cancer death rate for the weighted average of the "exposed" and "control" categories is 13.8 per 100,000. Over the 12 yr of the Garfinkel study, the actual rate averaged 13.3 per 100,000, a less than 4% difference. In other words, this analysis (Repace, 1984) appears to explain both the observed lung cancer death rate and observed risk-ratio of the Garfinkel ACS cohort.

Could this be due to chance? Suppose that, instead of 38% of women in the workforce, 100% of women lung cancer death rate would be 19.42, a 46% difference. Suppose 0% of women worked. Then the ratio of risks would be 1.26, a 5% difference from Garfinkel's result, but the lung cancer death rate would be 10.32 per 100,000, a 22% difference from Garfinkel's observation.

Suppose the exposure-response relationship of 0.6 LCDs/100,000 person-yr per mg/day yielded by extrapolation with the one-hit model from the risks in smokers is used. The lung cancer deaths per 100,000 contributed by passive smoking are then 0.27 (0.45 × 0.6) for the home and 1.1 (1.82 × 0.6) for the workplace. Application of these figures to the numbers of true and tainted controls and working and nonworking exposed women yields, after addition of the basal risk of 8.7 per 100,000, the figures shown in Table 6. The ratio of risks (all exposed:all controls) is then 1.03. Compared with the risk ratio in the Garfinkel (1981) study, this is a 14% difference. The lung cancer death rate for the weighted average of the "exposed" and "control" categories is 9.3 per 100,000, a 30% difference from Garfinkel's result.

When the one-hit model is used, the ratio of "all-exposed" to "true" controls is 1.09, a 38% difference with Hirayama's ratio. The corresponding lung cancer mortality rate is 9.45, a 39% difference with Hirayama's result.

Finally, using the phenomenological exposure-response relation, the ratio for "all exposed" and "true" controls is 1.7. Hirayama's (1981) average risk ratio was 1.78 from passive smoking, a 4.5% difference. Furthermore, if lung cancer risk rate calculation is performed with the tainted controls included as an exposed group, the result is 14.8 per 100,000, compared with Hirayama's observed 15.5 per 100,000, a 4% difference. In other words, the effect of moving the confounding tainted controls from Garfinkel's control group into his exposed group is to yield results within 5% of Hirayama's.

Thus, on the basis of this sensitivity analysis, it would appear that the phenomenological exposure-response relationship is better able to describe the results of the Garfinkel (1981) study than the one-hit model, and in addition, also appears to be able to explain quantitatively why the two large prospective studies of passive smoking and lung cancer yielded different results.

Table 5. Calculated lung cancer risks for each subgroup in the Garfinkel (1981) study using the 5 LCDs/100,000 person-yr/mg/day exposure-response relation.

Group	Rate
True controls	8.7
Tainted controls	17.8 (8.7 + 9.1)
All controls (weighted mean)	12.16
Exposed workers	20.05 (8.7 + 2.25 + 9.10)
Exposed nonworkers	10.95 (8.7 + 2.25)
All exposed (weighted mean)	14.41

Table 6. Calculated lung cancer risks for each subgroup in the Garfinkel (1981) study using the 0.6 LCDs per 100,000 person-yr/mg/day exposure-response relation.

Group	Rate
True controls	8.7
Tainted controls	9.8 (8.7 + 1.1)
All controls (weighted mean)	9.11
Exposed workers	10.07 (8.7 + 0.27 + 1.1)
Exposed nonworkers	8.97 (8.7 + 0.27)
All exposed (weighted mean)	9.39

Table 7. Comparison of estimated risks from various hazardous air pollutants. Risks have been assessed for non-occupational exposures of the general population to several hazardous air pollutants. All are airborne carcinogens; all but passive smoking are being regulated by society. The statistical mortality given is before control.

Pollutant	Estimated Annual Mortality[a,b]	Reference
Passive smoking	5000 LCDs per yr	(this work)
Vinyl chloride	<27 CDs per yr	(U.S. EPA, 1975)
Radionuclides (worldwide impact from Department of Energy facilities)	17 CDs per yr	(U.S. EPA, 1983b)
Coke oven emissions	<15 LCDs per yr	(U.S. EPA, 1984)
Benzene	<8 CDs per yr	(U.S. EPA, 1979b)
Arsenic	<5 LCDs per yr	(U.S. EPA, 1980)

[a]CD = cancer death; LCD = lung cancer death.
[b]Risks for passive smoking and radionuclides are best estimates, and risks for other pollutants are upper bound.

Comparison of the Estimated Risk of Passive Smoking with those of Hazardous Air Pollutants Currently Under Regulation

Although the quantitative estimates presented should be regarded as preliminary and subject to confirmation by further research, the evidence suggests that passive smoking appears to be responsible for about one-third of the annual lung cancer mortality among U.S. nonsmokers. To place these estimates in perspective, Table 7 gives a comparison of the estimated risk of passive smoking to risks estimated by the U.S. Environmental Protection Agency for the carcinogenic hazardous air pollutants currently regulated under section 112 of the Clean Air Act (SCEP, 1977). As Table 7 demonstrates, passive smoking appears to pose a public health risk larger than the hazardous air pollutants from all regulated industrial emissions combined.

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Appendix A: Modeling Exposure of Nonsmoking U.S. Adults to Ambient Tobacco Smoke

Introduction

Lifestyle is the integrated way of life of an individual; aspects of lifestyle which will be considered here have to do with the amount of time a nonsmoker spends in contact with smokers, and therefore with their effluent. Exposure of nonsmokers to tobacco smoke might be expected to be common in the United States because one out of three U.S. adults smokes cigarettes at the estimated rate of 32 per day (Repace and Lowrey, 1980), and an additional one out of six smokes cigars or pipes. Furthermore, indoor air pollution from tobacco smoke persists in indoor environments long after smoking ceases (Repace and Lowrey, 1980, 1982).

Earlier work (Repace and Lowrey, 1980) presented a model of nonsmokers' exposure to the particulate phase of ambient smoke which was supported by controlled experiments and field survey of the levels of respirable particles indoors and out, in both smokefree and smoky environments. This work, which established that ambient tobacco smoke imposed significant air pollution burdens on nonsmokers, was extended by later work (Repace and Lowrey, 1982) that further demonstrated the predictive power of this model. The model predicts a range of exposure of from 0 to 14 mg of cigarette aerosol per day, depending upon the nonsmoker's lifestyle. Exposures of prototypical nonsmokers were modeled, but no attempt was made to estimate the average population exposure. Concentrations of ambient tobacco smoke encountered by nonsmokers can be approximated by equilibrium values that are determined by the ratio of the average smoker density to the effective ventilation rate (Repace and Lowrey, 1980, 1982); in practice, design ventilation standards based on occupancy are useful surrogates for effective ventilation rates. On the average, a characteristic value of this ratio can be assigned to a particular microenvironmental class, e.g., homes, offices, restaurants, etc. (Repace *et al.*, 1980). Therefore, the average daily exposure of individuals can be estimated from the time-weighted sum of concentrations encountered in various microenvironments containing smoke (Ott, in press; NRC, 1981; Szalai, 1972; Repace *et al.*, 1980).

Exposure and lifestyle

It is important to realize that most persons' lifestyles are such that they spend nearly 90% of their time in just two microenvironmental classes, thus affording a great simplification of exposure modeling. Szalai (1972), as part of The Multinational Comparative Time Budget Research Project, which studied the habits of nearly 30,000 persons in 12 countries (from 1964 to 1966), has compiled data reporting the average time spent in various locations or microenvironments. The data for 44 cities in the United States, as analyzed by Ott (in press) are summarized in Table A1 (see also NRC, 1981).

Table A1 shows that U.S. urban people spend an average of 88% of their time in just two microenvironments: in homes and workplaces. Moreover, employed persons in the U.S. cities are estimated to spend only 3% of the day outdoors, while housewives spend only 2% outdoors (Ott, in press; NRC, 1981). Assume that these values are representative of the entire population. [In 1970, approximately three fourths of the population was urban (USDC, 1980).]

Table A1. Time spent in various microenvironments by persons in 44 U.S. cities, expressed in average hours per day. (Ott, in press; NRC, 1981; Szalai, 1972).

Microenvironment	Employed Men, All Days	Employed Women, All Days	Married Housewives, All Days
Inside one's home	13.4	15.4	20.5
Just outside one's home	0.2	0.0	0.1
At one's workplace	6.7	5.2	–
In transit	1.6	1.3	1.0
In other people's homes	0.5	0.7	0.8
In places of business	0.7	0.9	1.2
In restaurants and bars	0.4	0.2	0.1
In all other locations	0.5	0.3	0.3
Total	24.0	24.0	24.0

Modeling exposure of nonsmokers at work

Exposure of the population to the particulate phase of cigarette smoke can be modeled to determine both range of exposure and the nominal inhaled dose, which is exposure multiplied by the respiration rate (Altman and Dittmer, 1971).

Repace and Lowrey (1980, 1982a) have shown that the ambient concentration of tobacco smoke particles, Q, from cigarette smoking can be usefully represented by an equilibrium model of the form $Q = 650\ D_c/C$, where D_c is the number of burning cigarettes per 100m³, and C is the ventilatory air exchange rate in air changes per hour (ach). Rewriting this in terms of the occupancy of the space by habitual smokers (Repace and Lowrey, 1980) (for every 3 habitual smokers, there is one cigarette burning constantly), D_h($= 3D_c$):

$$Q = 217\ D_h/C, \quad (\mu g/m^3), \qquad (A1)$$

where D_h, is the habitual smoker density in units of smokers per 100 m³, and C, is the air change rate in units of air changes per hour (ach). Because The American Society of Heating, Refrigerating, and Ventilating Engineers (ASHRAE) (Leaderer *et al.*, 1981), sets consensus standards for ventilation rates in the United States, and because those standards are tied to expected building occupancy (e.g., ASHRAE, 1981), Eq. (A1) offers the possibility of modeling the range of nonsmokers' exposures by estimating the ranges of occupancy and air change rate. Appendix A1 estimates that the average annual exposure to ambient tobacco smoke particles by a typical nonsmoking U.S. worker is 1.8 mg/day, with an exposure probability of 63%.

Modeling exposure of nonsmokers at home

By reviewing data from time budget and census studies, the average length of time a person spends in the home microenvironment can be calculated. This time differs for gender and employment status. Taking into account the different amounts of time spent in the home by employed men, employed women, and homemakers, an estimate of occupancy-weighted average number of cigarettes smoked in the home during a 16-h waking day can be made. If the entire waking day is spent at home, 32 cigarettes per day (CPD) are smoked in the house by a smoker of either sex. An estimated occupancy-weighted average number cigarettes equal to 22 CPD smoked in the typical home is derived in Appendix A2. Using Eq. (A1), multiplied by the ratio 22/32, times a 1 m³/h respiration rate for a 16-h period, the calculation is made for a single-family detached dwelling of 340 m³ volume (see Appendix A3), assuming that on a 16-h basis, the entire finished volume of the home is available for dispersion of the smoke. A typical nonsmoker of either sex appears to be exposed to an average inhaled dose of 0.45 mg/day, assuming that occupancy of the home by smokers and nonsmokers is coincident.

Mean estimated dose to a typical adult from the most-frequented microenvironments

A probability-weighted average exposure to a hypothetical typical U.S. adult is estimated by combining the estimated dose to U.S. adults exposed in the workplace and at home, and by weighting the exposure received in each microenvironment by the probability of receiving it. Appendix A1 estimates that nonsmoking U.S. workers are exposed on the job to tobacco smoke with a probability of 63%. Appendix A2 estimates that nonsmoking U.S. adults are exposed at home to tobacco smoke with a probability of 62%. Table 1 (main text) gives the combinations of these probabilities, assuming that they are independent, i.e., that exposure at work is not correlated to exposure at home. Table 1 suggests that only a relatively small percentage (14%) of the population may escape daily passive smoke exposure. By contrast, individuals having exposure both at home and at work constitute a high exposure group, with the workplace likely contributing more exposure than the home by a ratio of 4 to 1.

On the basis of Table 1 it is estimated that the mean daily exposure of nonsmoking U.S. adults to tobacco tar and nicotine from the breathing of indoor air contaminated by cigarette smoke is about 1.43 mg/day, averaged over the two most-frequented microenvironments. This may be compared to the estimate of 14 mg/day to the hypothetical most-exposed individual (Repace and Lowrey, 1980). These results indicate that the typical U.S. "nonsmoker" appears to be exposed to a finite, non zero amount of tobacco aerosol, equivalent in value to three low-tar cigarettes (0.55 mg) per day.

In summation, it is possible, based on ASHRAE standards, time budget, and census surveys, the physics of indoor air pollution transport, and tables of respiration rates, to estimate the average exposure of a typical nonsmoking U.S. adult of working age. Using this methodology, estimates of the average exposure of the U.S. adult population of working age to the particulate phase of ambient tobacco smoke are made for the two most-

frequented microenvironments: the workplace and the home. It is estimated that 86% of adults of working age are exposed to ambient tobacco smoke on a daily basis, and 14% are not. It is estimated that the range of exposure varies from 0 to 14 mg of tobacco tar per day, and that the typical exposure, averaged over 100% of the population, is 1.43 mg/day. It also is estimated that those individuals who are exposed both at home and at work receive a daily average exposure of 2.4 mg/day, and that 39% of the adult worker population is in this category. Those individuals exposed *only* at home receive a daily exposure of 0.5 mg/day, and that 23% of the adult population is in this category. Finally, it is estimated that those individuals exposed *only* at work receive a daily exposure of 1.8 mg/day, and that 24% of the worker population is in this category. Thus these estimates suggest that the ratio of workplace dose to the exposure received at home is nearly 4:1, indicating that, on the average, the workplace is a more important source of exposure than the home environment. Consistency of these estimates of workplace and domestic exposure with field data is given in Appendices A1 and A2.

Appendix A1: Modeling the Average Daily Exposure to Cigarette Smoke for a Typical U.S. Nonsmoking Worker

It is possible to arrive at an estimated aggregate exposure because the range of occupancies (i.e., smoker densities) is tied to the range of ventilation rates, which in turn determine the range of concentration of ambient tobacco smoke to which nonsmokers are exposed. A form of Eq. (A1) is given that can be related directly to the ASHRAE Standards 62-73 (ASHRAE, 1973), promulgated in 1973, which set standards for natural and mechanical ventilation. The practical range of occupancy given in the ASHRAE Standard 62-1973 is from 5 persons/1000 ft² to 150 persons/1000 ft² (5.4 P/100 m² to 161 P/100 m²), for commercial and institutional buildings.

From 1946 to 1973, the operable engineering standard was descriptive of general practice rather than prescriptive: The American Standard Building Code Requirements for Light and Ventilation A53, Section 8 (ASA, 1946) described typical practice for mechanical ventilation based on floor area, not occupancy. Section 8 described minimum values of 0.5 CFM/ft² for offices, 1 to 1.5 CFM/ft² (4.4 to 6.6 L/s m²) for workrooms, and a range of 0.05 to 3 CFM/ft² (2.2 to 13.2 L/s m²) for public and institutional buildings, with the lower value applying to museums, and the upper value to dance halls. This implies air exchange rates varying from 3 to 18 ach, and at the maximum of 75% recirculation described, this range reduces to 0.75 to 4.5 ach. In 1970, 60.7% of the U.S. workforce worked in the white-collar and service occupations that inhabit such buildings (USDC, 1980). A 1979 survey of 3000 employers in large, medium, and small corporations indicated that smoking was prohibited in only 10.5% of white-collar workplaces and in 27.5% of blue-collar workplaces (NICSH, 1978). These percentages would likely have been less in 1970. Equation (A2) expresses the concentration, R, as a function of occupancy, which is now a surrogate (Repace and Lowrey, 1978, 1982a) for smoker density:

$$R = 25.6\, P_o/C_v \quad (\mu g/m^3), \qquad (A2)$$

where P_o is the occupancy in persons per 1000 ft² [100 m²], and C_v is the ventilatory air change rate in ach, as before. Exposures can be calculated by multiplying R by the integrated average respiration rate expected for an adult nonsmoker over an 8-h workday. A reasonable value is 8 m³ per workshift, a value corresponding to alternate sitting plus light work (Table A2). Multiplying Eq. (A2) by this rate yields the equation for the amount of tobacco tar inhaled, N_t:

$$N_t = 0.205\, P_o/C_v \quad (mg/8\ h), \qquad (A3)$$

where the other parameters are defined as in Eq. (A2). ASHRAE Standards 62-73 yield the ranges in P_o of 5 to 150 and in C_v of 0.15 to 18 ach.

Table A3 expresses the variation of these parameters from the absolute minimum airchange rate to the recommended minimum and maximum rates, and enables us to bound the modeled dose for the workplace. The extreme bounds of workplace exposure can be estimated to range from $1.35 \le N_t \le 6.77$ mg/8 h. This assumes that one-third of the occupants are smokers [following the U.S. average that one-third of the adult population smokes (USSG, 1979)], and that they smoke sales-weighted-average tar cigarettes at the rate of 32 per 16-h day (Repace and Lowrey, 1980). Clearly, the true minimum bound is zero, and the maximum exposure may be higher due to the presence of chainsmokers or a higher than average number of smokers, but what is desired is an expected average value for the workplace exposure. At the ASHRAE-recommended minimum ventilation, the upper bound for N_t will be 3.38 mg/8 h. Thus, the probable average range for N_t is between 1.35 and 3.38 mg/8 h. The average of these two figures, $N_t = 2.37$ mg/8 h, represents the mean exposure for U.S. workers who are on-the-job passive smokers. This value may be

Table A2. Range of typical adult respiration rates for different levels of effort after Altman and Dittmer (1971).

Activity Level	Respiration Rate (m³/h)
Resting	0.36
Sitting	0.60
Alternate Sitting & Light Work	0.99
Light Work	1.47
Heavy Work	2.04

Table A1. Calculation of the range of concentration Q, and exposure N_e to which nonsmokers are subject under the model given by Eqs. (A2) and (A3) assuming ASHRAE standard ventilation.

Occupancy	P_o [occupants per 1000 ft² (per 100 m²)]	C_r [airchanges/hour (ach)]	N_e Exposure (mg/8 h)	Q Concentration (μg/m³)
A. Using ASHRAE 62-73 recommended maximum makeup air based on occupancy				
Maximum	150	18	1.69	213
Minimum	5	0.5	2.03	256
Offices	10	1.5	1.35	170
B. Using ASHRAE 62-73 absolute minimum makeup air based on occupancy				
Maximum	150	4.5	6.77	853
Minimum	5	0.15	6.77	853
Offices	10	0.3	6.77	853
C. Using ASHRAE 62-73 recommended minimum makeup air based on occupancy				
Maximum	150	9	3.38	655
Minimum	5	0.3	3.38	655
Offices	10	0.9	2.26	284

transformed into a daily average using Table A1. In 1972, (USDC, 1980) 38% of the workforce was female: 0.38 × (5.2 workhours/day) and 62% was male: 0.62 × (8.7 workhours/day); the sum of these is 6.13 workhours/day, daily average. Thus, the daily average exposure is $N_e = (6.13/8) \times 2.37 = 1.82$ mg/day.

It now remains to estimate the percentage of workers who are exposed to cigarette smoke at work. The National Interagency Council on Smoking and Health conducted a survey of top management and health officials of 3000 U.S. corporations in 1978 (NICSH, 1978). A 29% response rate was achieved. The survey indicated that of blue-collar companies surveyed, 30.6% had no restrictions on smoking, 42% permitted smoking in designated areas, and 27.5% completely prohibited smoking. The corresponding percentages for the white-collar companies were, respectively, 74.3%, 15.2%, and 10.5%. Smaller companies were less likely to have restrictions. Among companies with restrictions, about one-half imposed penalties for violations. In addition, 65% of the respondents indicated that their policy was established after the release of the 1964 Surgeon General's Report on Smoking.

In 1970, white-collar workers constituted 48.3% of the workforce, blue-collar workers 35.3%, service workers 12.4%, and farmworkers 4% (USDC, 1980). The largest change in any category from 1960 to 1979 was that of white-collar workers, increasing by 7%. Since about one-half of the blue-collar companies imposed penalties for smoking, it will be assumed that 50% of the blue-collar nonsmokers were not exposed on the job. By contrast, it will be assumed that only 25% of the white-collar workers were not exposed. It will further be assumed that one-half of all workers follow white-collar smoking rules, and the other half, consisting of blue-collar workers, service workers, and farmworkers, follow blue-collar rules. Thus, the estimated weighted average percent of nonsmoking workers who are significantly exposed to tobacco smoke on the job is: 0.50 × 50% + 0.75 × 50% = 62.5%. By comparison, a 1983 survey of 1515 white-and blue-collar businesses sampled at random reported that "nearly two-thirds" had no smoking restrictions in the workplace (Tobacco Institute, 1984).

At this stage it must be asked whether the numbers calculated are reasonable in terms of measurements of ambient tobacco smoke under natural conditions. Repace and Lowrey (1980, 1982a), in a field survey of ambient tobacco smoke in 23 commercial buildings in the metropolitan Washington, D.C., area during 1979–1980, found concentrations ranging from about 100 μg/m³ to more than 1000 μg/m³. This range is quite compatible with the concentrations Q derived in Table A3. The average of all values measured under a variety of smoking conditions and ventilation rates by Repace and Lowrey was 242 μg/m³ (range 100 to 1000 μg/m³) for these 23 locations, corrected for background. This is compatible with the values calculated in Table A3. Breathing 242 μg/m³ of ambient tobacco smoke for 8 h at a rate of 0.99 m³ h yields an exposure of 1.92 mg/8 h or on a daily average basis, 1.92 × (6.13/8) = 1.47 mg.

In terms of relative exposures, these results also appear to be reasonable. In Appendix A2, an average smoking rate of 32 CPD was used (Repace and Lowrey, 1980). At current sales-weighted average tar plus nicotine values (14 mg) (USFTC, 1984), the typical smoker would inhale (14 mg/cig) × 32 CPD = 448 mg/day. In Table 1, the typical passive smoker was calculated to inhale 1.43 mg/day. This is a relative exposure ratio of 313:1. Wald *et al.* (1984a), in a study of urinary cotinine levels in smokers and nonsmokers, found the ratio (1645 ng/mL)/(6 ng/mL) = 274:1. Thus, the ratio of exposures calculated theoretically using the model derived here differs by only 14% from an experimentally derived value based on a biological marker of exposure.

Appendix A2. Calculation of the Estimated Daily Average Number of Cigarettes Smoked in the Average Home

Since the source strength depends upon the length of time smokers spend in indoor microenvironments, it is necessary to review pertinent information from time budget (Ott, in press; Szalai, 1972; NRC, 1981) and census (USDC, 1980) studies, which gives the average length of time that persons spend in various microenvironments.

From Table A1, it is seen that, allowing for 8 h of sleep, employed men spend 34.4% of the waking day in

the home; employed women spend 45.9% of the waking day in the home; "housewives" spend 81% of the waking day in the home. In 1979, approximately 42% of families with both the husband and wife present, both were employed. Thus for homes occupied by married couples, 66% of the waking day (weighted mean averaged over 42% working wives and 58% homemakers) the home is occupied by a wife, and 34% of the day, by a husband. If the average habitual smoker smokes 32 cigarettes per day (CPD), then the wife will smoke 21 CPD in the house, and the husband will smoke 11 CPD in the house.

Bonham and Wilson (1981) found that 62% of U.S. homes with children in 1970 contained one or more smokers, and 25% contained two or more. Thus we may assume that of homes with one or more smokers, 40% have two smokers, and 60% have one smoker. We have three cases to consider: (a) Husband and Wife Both Smoke, (b) Only Wife Smokes, and (c) Only Husband Smokes. In 40% of the smoking homes, case (a) is true, and in 60% of those homes, either cases (b) or (c) is true. Specifically, 38% of men and 30% of women smoke (US DHHS, 1979). Then the probability of case (c) being true is 34% (38/68 × 60%), and the probability of case (b) being true is 26% (30/68 × 60%). The weighted mean of these is given by the sum of the products of the percent of homes with a given number of smokers of either or both sexes, times the number of cigarettes per day smoked by either or both sexes: 0.40 × 32 + 0.26 × 21 + 0.34 × 11 = 22 CPD, estimated to be smoked daily in the average U.S. home, or about a pack per day. Is this theoretical estimate a reasonable number?

Dockery and Spengler (1981a, 1981b), in a 1-yr study of indoor air pollution in 68 homes in 6 U.S. cities, found that cigarette smoking was the dominant source of respirable particles (RSP); in a typical house in the study, the average 24-h RSP levels were increased by 0.88 $\mu g/m^3$ per cigarette smoked, and in a tightly sealed house, by a value of 2.11 $\mu g/m^3$. At an estimated occupancy-weighted average in-the-home smoking rate of 22 cigarettes per day, a 24-h average RSP level of about 19 $\mu g/m^3$ (22 CPD × 0.88 $\mu g/m^3$ CPD) is calculated for the typical house; in fact, Spengler *et al.* (quoted in NRC, 1981) observed a 24-h average of 19 $\mu g/m^3$ in 22 of the homes in the study where there was only 1 smoker. This number corresponds to an air exchange rate of 1.5 ach using the model (see Appendix A3). Since this air exchange rate is within the expected range (Repace and Lowrey, 1980, 1982), an average of 22 CPD smoked in the home provides a reasonable basis for estimating exposure.

The theoretical in-the-home number of cigarettes smoked in the home is weighted for occupancy during the waking day. Since there is no data differentiating occupancy for smokers and nonsmokers, it is assumed that the statistical occupancy of the nonsmoker is coincident with that of the smoker, i.e., that there is a nonsmoker present to receive the exposure. In order to calculate the daily dose received, Eq. (A1) is used with the parameters D_w = 0.29 smokers/100m³, C_r = 1.5 ach, times an occupancy factor of 22/32, times a respiration rate of 0.99 m³/h, times a 16-h maximum exposure day, yielding an estimated average exposure of 0.45 mg/day, for an adult nonsmoker, with an exposure probability of 62%.

A reasonable approximation to the probability of a typical nonsmoking adult being exposed to ambient tobacco smoke at home is 62%, the same as Bonham and Wilson (1981) above found for adults with children (in 1970, 56% of families had one or more children under 18) (USDC, 1980). No differentiation is made between male and female nonsmokers, since Friedman *et al.* (1983) observed that degree of passive smoking had little correlation with gender. In households with 2 or more smokers, there might not be an adult nonsmoker to be exposed; in this case, the probability of passive smoking (for a nonsmoker) reduces from 62% to 37%. However, the estimated total exposure (Table 1) only decreases by 8%, from 1.43 to 1.34 mg/day. In the absence of data on this point, it will be assumed that a nonsmoking adult is present.

Appendix A3. Calculation of the Ratio of the Habitual Smoker Density to the Air Exchange Rate for a Typical U.S. Single Family House

The typical range of annual closed-window air exchange rates in U.S. residences is generally considered to be of the order of 0.5 to 1.5 ach, with the range for the average residence of the order of 0.7 to 1.1 ach, and that of the tighter and newer residences of the order of 0.5 to 0.8 ach (Fuller, 1981). So-called energy-efficient structures have rates of the order of 0.3 to 0.5 ach. (Repace, 1982). A typical U.S. single-family detached house is estimated to have a floor area of 1500 ft.² [139 m²] with an 8-ft [2.4-m] ceiling, for a volume of 340 m³ (NAHB, 1981).

Thus, per habitual smoker, the ratio D_w/C_r = (1/3.4)/1.0 = 0.29 habitual smokers per hundred cubic meters per air change per hour. In 1978, nearly ⅔ of occupied housing units were single-family detached dwellings (USDC, 1980). It is assumed that the ratio calculated above is valid for multifamily dwellings as well (the volume of an apartment in a multifamily building is likely to be less, but the air exchange rate is likely to be greater).

Appendix B: Extrapolated Estimate of Risk From Passive Smoking

An alternative method of estimation of risk from passive smoking is calculated as follows. In 1980, 108,504 individuals in the United States were reported

to have died from lung cancer (USPHS, 1983). The 1982 Surgeon General's report on Smoking and Cancer estimated that 85% of LCDs are due to cigarette smoking (USSG, 1982); this yields 92,228 LCDs/yr. Lung cancers occur primarily in smokers over the age of 35 (NCI, 1966); in 1980, there were an estimated 29,225,000 smokers of all races and both sexes in this age bracket (USPHS, in press). It follows that in 1980, there were 3.156×10^{-3} LCDs per smoker of lung cancer age. In 1978 the average cigarette was 17 mg tar, and the average smoker smoked 32 cigarettes per day (Repace and Lowrey, 1980), for an estimated tar intake of 544 mg/day-smoker. (A 1980 lung cancer death reflects a 20- to 40-yr smoking history, during which smoking rates increased by, and tar levels decreased by, about 50% (USSG, 1979). Thus, 3.156×10^{-3} LCDs/smoker divided by 544 mg/day-smoker yields a rate of about 5.8×10^{-6} LCDs/yr per mg/day per smoker of lung cancer age.

Using a one-hit model (Hoel *et al.*, 1983; Crump, 1976) for the extrapolation of the risk from the estimated exposure of smokers down to the estimated exposure of nonsmokers provides an alternate exposure-response relationship. Crouch and Wilson (1981) have used this model, which saturates at high exposures, but which is linear at low exposures. This model has the form $P(D) = 1 - \exp(bD)$, where $P(D)$ is the estimated risk, b is the exposure-response function, and D is the exposure. The one-hit model, because of its functional form, can be considered as the first stage of the more complex multistage model. (U.S. EPA, 1983a; Hoel *et al.*, 1983) Whenever the data can be fitted adequately by the one-hit model, estimates of both models will be comparable (U.S. EPA, 1983a; Crump, 1976; Hoel *et al.*, 1983).

From above, $b = 5.8 \times 10^{-6}$ LCDs per year per mg/day. $D = 1.5$ mg/day, from the estimated average exposure for the typical U.S. nonsmoker (Appendix A), assuming that per milligram, tobacco tar produces the same carcinogenic response in nonsmokers as it does in smokers. This calculation yields an estimated annual LCD risk about 0.87×10^{-5} from passive smoking, or about an order of magnitude lower than the phenomenological estimate made earlier. In this exposure range, this result is essentially the same as would be obtained from a linear extrapolation.

The primary age group at risk of lung cancer is that ≥ 35 yr (Reif, 1981a, 1981b). Therefore, in the calculation that follows only nonsmokers ≥ 35 yr will be assumed to be at risk of lung cancer. In 1980, there were about 63.8 million nonsmokers aged ≥ 35 (USPHS, in press). Thus, the alternative risk estimate is derived from multiplying 0.87 LCDs/yr per 100,000 passive smokers times 63.8×10^5 passive smokers at risk, yielding 555 LCDs per year in U.S. nonsmokers from passive smoking, using the one-hit model of carcinogenesis for extrapolation.

Appendix C: Age-Standardized Calculation of Estimated Annual U.S. Mortality and Loss of Life Expectancy From Involuntary Exposure to Ambient Tobacco Smoke

Approximately 50% of SDAs in the cancer age range (>35 yr old) are adult converts to the church; others were either born into an SDA home or joined the church prior to age 20, typically with other immediate family members. A large proportion of SDAs tend to be heavily involved in church activities. Only a small proportion of SDAs report current use of cigarettes (males, 1.7%; females 0.5%) (Phillips *et al.*, 1980b). (By contrast, in 1970, 43.5% of adult males and 31.1% of adult females in the general population aged ≥ 17 yr reported smoking) (USDHHS, 1979).

Moreover, a substantial portion of SDAs work for "an organization owned and operated by the SDA Church" [nearly 45% of SDA females and 40% of SDA males in the study group (aged ≥ 25 yr), reported working for the SDA Church.] (Phillips *et al.*, 1980a, 1980b). Clearly, SDAs are less likely than the general population to be involuntarily exposed to tobacco smoke, as children or as adults, at home or in the workplace, because neither SDA homes nor SDA businesses are likely to be places where smoking is permitted, and because the great majority of SDA family and social contacts are among other SDAs who do not smoke (Phillips *et al.*, 1980b).

Table C1 shows the age-standardized calculation of estimated loss of life expectancy and annual lung cancer mortality from passive smoking. The calculation is based on the lung cancer mortality difference between two Southern California cohorts of self-reported nonsmokers who never smoked. Based on lifestyle differences, they appear to have different average levels of involuntary smoke exposure. The more-exposed group are designated non-SDAs, and the less-exposed group SDAs (see main text).

Columns 1, 2, 5, and 6 are tabulations from which age-adjusted mortality rates were calculated in the study of mortality in the Seventh-Day Adventist (SDA) by Phillips *et al.* (1980a, 1980b). Columns 1 and 2 and 5 and 6 give the age-specific lung cancer deaths and person-years at risk, respectively, for the SDA and the non-SDA. The fractional number of LCDs in column 1 is due to a correction for out-migration of the SDA population from the study area.

Columns 3, 7, 10, and 11 show the average numbers of individuals at risk annually during the study, allowing for those who died during the study. Columns 4 and 8 show the annual average lung cancer death rate (LCD) per 100,000 persons, and column 9 gives the differences between the non-SDAs and SDAs in those rates. Column 12 gives average LCD rates weighted to reflect the fact that there were three times as many women as men in the study, and that the female data attained statistical significance whereas the male did not—although the

combined data were significant (Phillips *et al.*, 1980a, 1980b). A common LCD rate is assumed for both sexes in the calculation that follows. Also, it will be assumed that the entire LCD rate difference is due to passive smoking (see discussion on confounding factors in Appendix D).

Next, this calculation will be extrapolated to the entire U.S. nonsmoking population aged ≥ 35 yr. Column 13 gives the mean age of the individuals in the 5-yr age group, and column 14 gives the number of persons alive at that mean age per 100,000 born alive. Column 15 gives the total number of persons in the 5-yr age group (5 × column 14) per 100,000 born alive (whites only) from the 1974 U.S. Life Tables (USDHHS, 1975). Column 16 gives the age-specific LCD rates attributed to passive smoking, standardized to (i.e., weighted by) the age specific population distribution in 1974 for U.S. whites (column 12 times column 15).

Column 17 gives the average life expectancy corresponding to the mean age given in column 13, which is taken to represent that of the entire 5-yr age group. Column 18, the product of columns 16 and 17, gives the estimated age-specific age-standardized person-years of life lost due to lung cancer from passive smoking.

The sum of the values of column 18 gives an estimated 3932 person-yr of life lost due to passive smoking per 100,000 persons alive at age 35 in the U.S. population in 1979. 3932 person-yr, when divided by the 94,724 persons (USDHHS, 1975) at risk at age 40 (LCDs were not observed at earlier ages in the SDA study; however, they are observed in the general nonsmoking U.S. population at age 35) (USSG, 1979) yields 15 days, the mean number of days of life lost, and, multiplying by the peak-to-mean exposure ratio, 148 days for the maximum number of days lost (where the risks of the non-white population are taken to be the same as for the white population.)

Column 19 is column 16 times 62.424 million divided by the sum of column 15. The sum of column 19 gives an estimated age-standardized mortality total of 4,665 LCDs per year in U.S. nonsmokers from passive smoking (where there were 93,636,000 persons aged ≥ 35 yr

Table C1. Age-standardized estimation of lung cancer deaths from passive smoking.

Females

5 yr. Age Group	SDA Never Smokers 1. Total LCDs (17 yr period)	2. Person yr at Risk	3. Average Annual No. of Persons	4. LCDs per 100,000 Person Years	Non-SDA Never Smokers 5. Total LCDs (12.58 yr period)	6. Person yrs. at Risk	7. Average Annual No. of Persons at Risk	8 LCDs per 100,000 Person Yrs
35-39	0	3791	223.0	0	0	5766	458.3	0
40-44	0	11494	676.1	0	1	16466	1308.3	6.0731
45-49	0	18757.5	1103.4	0	2	38319	3046.9	5.2193
50-54	1.119	24808.5	1459.3	4.5106	4	61630	4899.0	6.4959
55-59	1.000	24702	1453.1	4.0483	8	71289	5666.9	11.222
60-64	1.101	24051.5	1414.8	4.5777	7	65054	5171.2	10.760
65-69	1.148	23326.5	1372.1	4.9214	4	55614	4420.8	7.1924
70-74	0	21809	1282.9	0	9	44248	3517.3	20.340
75-79	1.000	18822	1107.2	5.3219	10	29250	2325.1	34.184
80-84	7.775	13435.5	790.3	57.869	6	15301	1216.3	39.213
85+	2.258	10017.5	589.5	22.541	10	7891	627.3	126.73
Total	15.401	195,015	11,472	103.7899	61	410,828	32,657	267.428

Males

5 yr. Age Group	SDA Never Smokers				Non-SDA Never Smokers			
35-39	0	1926.5	113.0	0	0	1581	119.3	0
40-44	0	5732.5	337.2	0	0	3479	276.6	0
45-49	0	9177	539.8	0	0	9662	768.0	0
50-54	0	11480	675.3	0	1	19313	1535.2	5.1779
55-59	1.119	10359.5	609.4	10.8017	2	23848	1895.6	8.3863
60-64	1.000	8763.5	515.5	11.440	4	19535	1552.9	20.4761
65-69	3.401	7386.5	434.5	46.0435	8	14105	1121.2	56.7173
70-74	1.115	6360.5	374.1	17.5301	0	9786	777.9	0
75-79	0	5278.5	310.5	0	2	6541	520.0	30.5764
80-84	1.143	3957.0	232.8	28.8855	4	3517	279.6	113.733
85+	2.235	3160.0	185.9	70.7278	2	1671	132.8	119.669
Total	10.013	73581.5	4328.0	185.4266	23	113,038	8979.1	354.7564

Table C1. (Continued).

5 yr Age Group	Females 9. Annual LCDs per 100,000 (Non-SDA)-(SDA)	Females 10. Average Age-Specific No. of SDA & Non SDA at Risk	Male/Female 11. Average Age-Specific No. of SDA & Non SDA at Risk	Male/Female 12. Annual Weighted Mean LCD per 100,000 (unisex)	13. Mean age of 5 yr Group	14. Mean No. of Persons At Risk at each yr of 5 yr Group	15. Mean No. of Persons At Risk in Entire 5 yr Age Group	16. Age Specific Age Stand (1974 U.S. White Population) LCDs
35–39	0	681	913	0	37.5	95,201	476,005	0
40–44	6.0731	1985	2599	4.64	42.5	94,122	470,610	21.84
45–49	5.2193	4149	5457	4.50	47.5	92,339	461,645	20.77
50–54	1.9803	6358	8569	2.81	52.5	89,590	447,950	12.59
55–59	7.1737	7120	9625	4.67	57.5	85,477	427,385	19.96
60–64	6.1823	6586	8654	6.87	62.5	79,396	396,980	27.27
65–69	2.2710	5793	7349	4.05	67.5	71,177	355,885	14.41
70–74	20.340	4800	5952	13.0	72.5	60,455	302,275	39.30
75–79	28.875	3432	4263	29.2	77.5	46,689	233,445	68.17
80–84	−18.656	2006	2518	2.35	82.5	31,209	156,045	3.67
85+	104.189	1217	1536	92.6	87.5	11,913	59,565	55.16
Total	163.6477	44,127	57,435	164.69	(1974 Census (whites per 100,000 at Birth)		3,787,790	203.14

5 yr. Age	Males	
35–39	0	232
40–44	0	614
45–49	0	1308
50–54	5.1779	2211
55–59	−2.4152	2505
60–64	9.0651	2068
65–69	10.6740	1556
70–74	−17.5301	1152
75–79	30.5764	831
80–84	84.8475	512
85+	48.9612	319
Total	169.3568	13,308

Table C1. (Continued).

5 Yr. Age Group	17. Average life Expectancy for the 5-year Age Group	18. Person years of life lost due to LCDs from passive smoking	19. LCDs per year in age group in entire 1979 U.S. nonsmoking population aged ≥ 35 yr
35–39	–	0	0
40–44	33.1	723	360
45–49	28.7	596	343
50–54	24.6	310	207
55–59	20.8	415	329
60–64	17.2	469	449
65–69	14.0	202	237
70–74	11.1	436	647
75–79	8.6	586	1123
80–84	6.6	24	61
85+	3.1	171	909
	17 ± 9	3932	4665

in 1979, and two-thirds or 62,424,000 of these were nonsmokers).

Examining column 19, shows that of those individuals assumed to contract lung cancer from passive smoking, approximately 1½% do so at each year of age from 40 to 69, and over age 70, approximately 3% do so each year. Of those who actually contract fatal lung cancer from passive smoking, the mean life expectancy lost is about 17 ±9 yr, and about 8% lose as much as 33 yr.

Appendix D: Discussion of Confounding Factors

The IARC criteria for causality and human cancer specify that possible sources of bias and confounding error should be considered (IARC, 1979). What factors other than passive smoking could account for a lung cancer difference between two cohorts?

The most obvious one is misclassification. Some of the individuals classified as nonsmokers could have

been smokers or exsmokers, giving rise to a spurious effect. Workplace or residential exposure to lung carcinogens or dietary differences between the cohorts might also give rise to spurious differences. However, this is not likely to be an effect constant over 14 positive studies in six different countries, all of which report about a doubling of risk when the exposure variable is spouses' smoking.

Arsenic, asbestos, beryllium, chloroethers, chromium, coke oven emissions, nickel, radon, and vinyl chloride, as well as tobacco smoke, have been implicated in the etiology of lung cancer (Ives, 1983; Selikoff, 1981). Possible differences due to industrial exposures should be expected primarily in blue-collar workers. Phillips *et al.* (1980a, 1980b) have stated that the SDA/non-SDA subgroups were demographically and educationally similar, suggesting similar occupational distributions, although there is no information on this point. There is no reason to believe that domestic radon levels, which are a property of the soil, would be any different in SDA homes than non-SDA homes. Finally, it should be considered that co-exposures to other lung carcinogens (e.g., radon) may increase the effect of passive smoking (Bergman and Axelson, 1983).

It is also possible that dietary differences between the two groups might have contributed to the SDA/non-SDA lung cancer difference. For example, 54% of SDAs follow a lacto-ovarian diet and 41% rarely use caffeine beverages. However, Hirayama (1981a, 1981b, 1983a, 1983b) observed a dose-response relationship between exposure to passive smoking and lung cancer even in those with an apparently cancer-inhibiting diet. Also, SDA/non-SDA cancer differences are not significant for other smoking-related cancer sites; this runs counter to a protective effect of diet as a confounding factor. Finally, Hirayama (1983a) observed that the magnitude of this effect varied from a mortality ratio of 1 for passive smoking women who did not follow a protective diet to 0.82 for women who use green-yellow vegetables only occasionally, to 0.72 for women who ate them daily. Thus the magnitude of the effect does not appear to be sufficient to account for the observed SDA/non-SDA lung cancer difference. Moreover, if 40% of the SDAs work for church-run organizations, 60% do not; these 60% surely must be subject to some passive smoking in the workplace, at least partially offsetting the effects of potential dietary or occupational differences with the non-SDAs.

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